CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS

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CERTIFICATE

This is to certify that the dissertation titled **"CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS"** is the bona fide original work of DR.N.VIJAY ANAND in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2007. The Period of study was from august 2005 to may 2006.

PROF. S. NATARAJAN, M.D.
Professor and Head of the
Department Of Medicine,
Govt. Stanley Medical College
And Hospital,
Chennai – 600 001.

PROF .V.RUKMANI, M.D.
Addl. Professor of Medicine,
Govt. Stanley Medical College
and Hospital,
Chennai – 600 001.

Dr. D.R.GUNASEKARAN, M.S., F.I.C.S.
DEAN
Govt. Stanley Medical College & Hospital,
Chennai – 600 001.

DECLARATION

I, **DR.N.VIJAY ANAND,** solemnly declare that dissertation titled **"CLINICAL OUTCOME OF STROKE IN RELATION TO ADMISSION DAY GLYCEMIC STATUS"** is a bona fide work done by me at Govt .Stanley Medical College and Hospital from august 2005 to may 2006 under the guidance and supervision of my unit chief **PROF. V.RUKMANI,M.D.,** Addl. Professor of Medicine.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

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Date :

DR. N. VIJAY ANAND

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1. INTRODUCTION

Among all the neurological diseases of adult life, Cerebro vascular accidents clearly ranks first in frequency of importance. At least fifty percent of neurological diseases in general hospital are due to stroke. Cerebro vascular accident includes ischemic stroke, hemorrhagic stroke, and cerebro vascular anomalies such as intracranial aneurysm, AV malformation and cortical venous thrombosis. Stroke, after heart disease and cancer, is the third most common cause of death[1] .With the introduction of effective treatment for hypertension, there has been a marked reduction in the frequency of stroke.

Diabetes mellitus by virtue of its association with micro vascular and macrovascular[2] disease is an important risk factor in the genesis of stroke. Most of the diabetic patients with stroke have raised glycosylated hemoglobin indicating that most of them have uncontrolled diabetes. Diabetics and stress Hyperglycemics have severe strokes resulting in poor outcome. Stroke is twice[3] more common in diabetics than in non diabetics. Hypertension is common in diabetes and accelerates atherosclerosis which promotes intracranial small vessel disease and heart disease leading to lacunar and embolic infarction respectively. There are several risk factors that determine the outcome of stroke. Hyperglycemia, fever , neuroprotective agents are those which are widely studied.

2. AIM OF THE STUDY

To measure the blood glucose level within twenty four hours of the onset of stoke in both diabetics and in non diabetics and to evaluate the severity and prognosis in both diabetics and non diabetics in relation to hyperglycemia.

3. REVIEW OF LITERATURE

DEFINITION :

Stroke (cerebro vascular accident) is a rapidly developing episode of focal and at times global loss of cerebral function with symptoms lasting more than 24hours or leading to death with no apparent cause other than that of vascular origin. [4]

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Transient ischemic attack (TIA) is an acute loss of focal cerebral or monocular function with symptoms lasting less than 24hours.

Reversible ischemic neurological deficit (RIND) refers to neurological deficit that disappears within 7 days of onset.

EPIDEMIOLOGY

RISK FACTORS [5]

NON-MODIFIABLE RISK FACTORS

Age : Increasing age is most powerful risk factor for cerebral infarction , intracerebral hemorrhage and subarachnoid hemorrhage as well as TIA

Sex : men are more at risk for ischemic stroke than woman up to 75 years of age.

Race : the risk is more in Blacks than Whites

Family history : there is an increased incidence of stroke in individuals who have a first degree relative affected with stroke or who have paternal or maternal history of death or disability due to stroke

MODIFIABLE RISK FACTORS [5]

Hypertension : It is an important predisposing factor not only for cerebral hemorrhage but also for infarction. Both systolic and diastolic pressures contribute to the risk though there is no critical level above which it operates. The reduction of BP by 10-12mmHg systolic and 5-6mmHg diastolic was found to be associated with 38% reduction in stroke incidence.

Diabetes mellitus : It is an independent risk factor for stroke. It increases the susceptibility to coronary , femoral , and cerebral atherosclerosis. The relative risk increases two to fourfold in diabetics patients with diabetic complications like retinopathy and autonomic neuropathy have a higher incidence of ischemic stroke

Hyperglycemia And Stroke :

Elevated blood glucose is common in the early phase of stroke. The prevalence of hyperglycemia, defined as blood glucose level more than 6.1mmol/l has been observed in two third of all ischemic stroke subtypes on admission and in at least fifty percent in each subtype including lacunar stroke[6]. Extensive experimental evidence in stroke models support that association between blood

glucose and functional outcome has been found in increasing number of clinical studies. Although no intervention stroke studies have addressed the reversal of hyperglycemia, active lowering of elevated blood glucose by rapidly acting insulin is recommended in most published guidelines , even in non diabetic patients (European Stroke Initiative, EUSI, guidelines > 10 mmol/l ; American Stroke Association, ASA, guidelines > 300 mg/dl) [7].

Causes of Acute Hyperglycemia :

Although up to one third of acutely stroke patients have either diagnosed or newly diabetic, probably a major proportion of patients have stress hyperglycemia, mediated partly by the release of cortisol and nor epinephrine. It is also a manifestation of relative insulin deficiency, which is associated with increased lipolysis even in non diabetic patients; stress hyperglycemia may be a marker of glucose regulation in individuals with insulin resistance and developing diabetes.

Hypercholesterolemia : It is a secondary risk factor indirectly affecting the risk of stroke. Reducing high cholesterol levels decreases the incidence of coronary artery disease which is the main cause of mortality in patients with cerebro vascular disease.

Non valvular atrial fibrillation : It is common in the age group of 65 – 85years and attributes to a five fold increase in the incidence of

embolic stroke especially in those patients who have recent CCF , arterial hypertension and prior thrombo embolism.

Cigarette smoking : It increases the risk of infarction in men and women by predisposing to carotid atherosclerosis. Smokers experience a reduction in stroke risk only after 5 years following cessation of smoking.

Alcohol : Light to moderate intake reduces the risk by increasing the HDL concentration, whereas heavy drinking increases the risk. More than one drink per day for women and more than two per day for men increases the BP, obesity and triglycerides level thereby increasing the risk.

TIA : Three times greater risk for subsequent stroke or death due to vascular causes ,especially those with hemispherical TIA. Patients with a previous history of stroke are at risk for a subsequent stroke. The risk of stroke recurrence is increased by the presence of dementia.

Carotid artery stenosis : Asymptomatic Carotid artery stenosis less than 75% carries a risk of 1.3% annually whereas risk increases to 10.5% per year if the stenosis is greater than 75%. Ulcerated .echoluscent and heterogeneous plaques with a soft-core are at higher risk for embolism.

Blood factors : Elevated haematocrit , hemoglobin and viscosity of the blood is associated with increased risk of ischemic stroke. Higher mean levels of plasma fibrinogen, factor VIII , von willebrand's factor, anti-thrombin III and lower mean levels of protein C is associated with increased risk. The

Antiphospholipid antibodies is associated with increased risk of ischemic stroke. Serum folate concentration less than or equal to 9.2 nmol/L alone may be risk factor for ischemic stroke.

Hormones : High dose estrogen oral contraceptives increases the risk of stroke in young women , while postmenopausal estrogen supplementation reduces the risk. The risk of cerebral infarction is increased in the first 6 weeks following delivery.

Others : Abdominal or truncal obesity, physical inactivity, habitual snoring are associated with increased risk of ischemic stroke.

PATHOPHYSIOLOGY

Stroke is 90% ischemic and 10 % hemorrhagic[8] . Abrupt disruption of focal cerebral blood flow causes acute ischemic stroke .The causes of decreased cerebral blood flow includes abrupt occlusion of small penetrating arteries arterioles, single or multiple arterial stenosis, arteritis, arterial dissection, venous occlusion, and profound anemia.

When cerebral blood flow falls below a critical value of 20ml /100g/min there is a loss of neuronal electrical function which is a reversible. When cerebral blood flow falls below a critical value of 10ml / 100g/min then aerobic mitochondrial metabolism fails and anaerobic metabolism leads to lactic acidosis.

As a sequel to this sodium and water enters the cell and potassium

leaks out of the cell due to failure of energy dependent intracellular homoeostasis leading to irreversible cell death.

Based on these facts the concept of ischemic penumbra was formulated. It is an area of brain that has reached the reversible stage of electrical failure but has not yet passed into the irreversible stage. Thrombolytic agents are used in this time window to salvage the ischemic penumbra zone.

HOW ELEVATED GLUCOSE INJURES THE ISCHEMIC BRAIN

By provoking anaerobic metabolism, lactic acidosis and free radical production, hyperglycemia may exert direct membrane lipid per oxidation and cell lysis in metabolically challenged tissues. Moderately and severely increased blood glucose has been found to further the metabolic state and mitochondrial function in the area and ischemic penumbra[9]. Insulin resistance is a known risk factor for the onset of stroke acting through a number of intermediate vascular disease risk factors (i.e. thrombophilia, endothelial dysfunction and inflammation)[10]. The evolution of acute infarction may be explained by the very same vascular factors, explaining why ischemic time seems to fly faster in patients with diabetes or grave hyperglycemia. Relative insulin deficiency liberates circulating free fatty acids, which together with hyperglycemia, diminishes vascular reactivity[11,12]. Furthermore, lowering glucose with insulin has been reported to reduce ischemic brain damage in an animal model[13].

The evolution of an infarction is accompanied by glutamate release

mediating repeated waves of spreading depression (SD) , another mechanism believed to propagate the necrosis of penumbral tissues. Although hyperglycemia alone did not trigger early response genes in cortical tissues of rats, in conjunction with induced SD, the expression of c-fos and cox-2 were substantially increased[14]. This suggested that increased glucose may trigger untoward intracellular biochemical cascades also by altering early gene expression in metabolically challenged neurons.

The blood brain barrier is well known to be vulnerable to hyperglycemia, presumably through the liberation of lactic acid and free radicals. The recent experimental study by Song et al in a rat model of collagenase induced intra cerebral hemorrhage add that hyperglycemia aggravates edema formation in zone surrounding the hemorrhage[15]. The study also documented increased cell death measured by TUNNEL staining. It is conceivable that hemorrhages are surrounded by a one of similarly challenged tissues as infarctions are where the availability of glucose influences the metabolic state.

COMMON CAUSES OF ISCHEMIC STROKE :

1.Thrombosis

Lacunar stroke (small vessel)

Large vessel thrombosis

Dehydration

2.Embolic occlusion

A. Artery-to-artery

Carotid bifurcation

Aortic arch, Arterial dissection

B. Cardio embolic

Atrial fibrillation

Mural thrombus

Myocardial infarction

Dilated cardiomyopathy

Valvular lesions

Mitral stenosis

Mechanical valve

Bacterial endocarditis

Paradoxical embolus

Atrial septal defect

Patent foramen ovale

Atrial septal aneurysm

Spontaneous echo contrast

UNCOMMON CAUSES :

1. Hypercoagulable disorders

Protein C deficiency

Protein S deficiency

Anti thrombin III deficiency

Antiphospholipid syndrome

Factor V Leiden mutation

Prothrombin G20210 mutation

Systemic malignancy

Sickle cell anemia

Beta Thalassemia

Polycythemia Vera

Systemic lupus erythematosus

Homocysteinemia

Thrombotic thrombocytopenic purpura

Disseminated intravascular coagulation

Dysproteinemias

Nephrotic syndrome

Inflammatory bowel disease

Oral contraceptives

2. Venous sinus thrombosis

3. Fibro muscular dysplasia

4. Vasculitis Systemic vasculitis

(PAN, Wegner's, Takayasu's, giant cell arteritis)

Primary CNS vasculitis

Meningitis (syphilis, tuberculosis, fungal, bacterial, zoster)

5. Cardiogenic

Mitral valve calcification

Atrial myxoma

Intra cardiac tumor

Marantic endocarditis

Libman-Sacks endocarditis

6. Subarachnoid hemorrhage vasospasm

7. Drugs: cocaine, amphetamine

8. Moyamoya disease

9. Eclampsia.

CLINICAL SYNDROMES [5]

TRANSIENT ISCHAEMIC ATTACKS

TIAs are abrupt in onset , brief in duration and recovery is possible in 24 hours . Recurrent TIAs can occur. Recognition and treatment is important as a complete stroke can be prevented.

Carotid TIAs

These are characterized by monocular blindness with recovery within Few minutes, visual field disturbance in the form of transient hemianopia and

speech disturbance due to dominant hemispherical dysfunction. Hemi paresis and hemi sensory loss can occur both in vertebro basilar TIAs as well as carotid TIAs.

Vertebro Basilar TIAs

These are characterized by prominent visual symptoms like Diplopia, homonymous hemianopia and cortical blindness, transient vertigo dizziness, unsteadiness due to cerebellar dysfunction and transient lower cranial nerve symptoms like dysarthria, perioral numbness, nasal regurgitation.

Drop attacks occur without any warning .It is thought to be due to ischemia of relays in reticular systems which normally function as part of reflex antigravity mechanism.

Subclavian steal

This syndrome occurs when there is occlusion of the Subclavian artery proximal to origin of vertebral artery which results in retrograde flow of blood down the vertebral artery during exercising the arm thereby leading on to symptoms of hindbrain ischemia.

Mini Strokes

Mini strokes are characterized by episodic ischemic symptoms which recover within 24 hours but there are small infarcts or hemorrhage in CT and are as significant as the TIAs

Stroke in evolution

Stroke in evolution refers to the slow progression of neurological deficit over several hours.

Major strokes

Major stroke are of sudden onset with loss of consciousness at the onset or soon after. It is very difficult to distinguish clinically between infarction or hemorrhage. Headache and vomiting if present, usually denotes hemorrhage especially if it is associated with rapid loss of consciousness. Athero –thrombo embolism is suggested by the presence of bruit over the carotid arteries.

Unusual types of stroke

Multi-infarct dementia, watershed infarction.

Classification of stroke on the basis of oxford shire community stroke

Sub classification [16]

Total anterior circulation syndrome (TACS)

Implies a large cortical stroke in middle cerebral or middle and anterior cerebral artery territories. it is characterized by a combination of

- new higher cerebral dysfunction
- Homonymous visual field defect
- an ipsilateral motor and / or sensory deficit involving at least

two out of three areas of the face , arm or leg

Partial anterior circulation syndrome

Implies a cortical stroke in middle or anterior cerebral arterial territory . This includes patients with two out of three components of the Total anterior circulation syndromes or new higher cerebral dysfunction alone or motor / sensory deficit more restricted than those classified as a TACS .

Lacunar syndrome

Implies a sub cortical stroke due to a small vessel disease

- Pure motor stroke
- Pure sensory stroke
- sensory motor stroke
- ataxic hemi paresis

Evidence of higher cortical dysfunction or disturbance of consciousness excludes Lacunar syndrome.

posterior circulation syndrome

1. Ipsilateral cranial nerve palsy with contra lateral motor and sensory deficit,

2. Bilateral motor and or sensory deficit

3. Disorder of conjugate eye movement

4. Cerebellar dysfunction without ipsilateral long tract involvement

5. Isolated homonymous visual field defects

ANTERIOR CEREBRAL CIRCULATION SYNDROME

no	Signs and symptoms	Structures involved
1.	Paralysis of opposite foot and leg	Motor leg area
2.	Lesser degree of paresis of opposite arm	Involvement of cortical / corona radiata of arm fibers
3.	Cortical sensory loss over toes, foot and leg	Sensory area for foot and leg
4.	Urinary incontinence	Sensory motor area in Para central lobule
5.	Contralateral grasp reflex,suckling reflex, gegenhalten	Medial surface of the posterior frontal lobe
6.	Abulia,slowness,delay,intermittentinterruption, lack of spontaneous whispering, reflex distraction to sights and sounds	Cingulate gyrus, medial inferior portion of frontal, parietal and temporal lobes
7.	Impairment of gait and stance (gait apraxia)	Frontal cortex near leg motor area
8.	Dyspraxia of left limbs, Tactile aphasia in left limbs	Corpus callosum

MIDDLE CEREBRAL CIRCULATION SYNDROME

no	Signs and symptoms	Structures involved
1	Paralysis of the contra lateral face, arm, and leg; sensory impairment over the same area (pinprick, cotton touch, vibration, position, two-point discrimination, stereognosis, tactile localization)	Somatic motor area for face and arm and the fibers descending from the leg area to enter the corona radiata and corresponding somatic sensory system
2	Motor aphasia	Motor speech area of the dominant hemisphere
3	Central aphasia, word deafness, anomia, jargon speech, sensory agraphia, acalculia, alexia, finger agnosia, right-left confusion (the last four comprise the Gerstmann syndrome	Central, suprasylvian speech area and parieto occipital cortex of the dominant hemisphere
4	Conduction aphasia	Central speech area (parietal operculum)
5	Apractognosia of the minor hemisphere (amorphosynthesis), anosognosia, hemiasomatognosia, unilateral neglect, agnosia for the left half of external space, dressing "apraxia," constructional "apraxia," distortion of visual coordinates, inaccurate localization in the half field, impaired ability to judge distance, upside-down reading, visual illusions (e.g., it may appear that another person walks through a table)	Non dominant parietal lobe (area corresponding to speech area in dominant hemisphere); loss of topographic memory is usually due to a non dominant lesion, occasionally to a dominant one)
6	Homonymous hemianopia (often homonymous inferior quadrantanopia)	Optic radiation deep to second temporal convolution
7	Paralysis of conjugate gaze to the opposite side	Frontal contraversive field or projecting fibers

POSTERIOR CEREBRAL CIRCULATION SYNDROME

Peripheral territory

no	Signs and symptoms	Structures involved
1	Homonymous hemianopia (often upper quadrantanopia)	Calcarine cortex or optic radiation
2	Bilateral homonymous hemianopia, cortical blindness, awareness or denial of blindness; tactile naming, achromatopia (color blindness), failure to see to-and-fro movements, inability to perceive objects not centrally located, apraxia of ocular movements, inability to count or enumerate objects, tendency to run into things that the patient sees and tries to avoid	Bilateral occipital lobe with possibly the parietal lobe involved
4	Verbal dyslexia without agraphia, color anomia	Dominant calcarine lesion and posterior part of corpus callosum.
5	Memory defect	Hippocampal lesion bilaterally or on the dominant side only
6	Topographic disorientation and prosopagnosia	Non dominant, calcarine, and lingual gyrus.
7	Simultagnosia, hemi visual neglect	Dominant visual cortex, contra lateral hemisphere
8	Unformed visual hallucinations, peduncular hallucinosis, metamorphosis, teleopsia, illusory visual spread, palinopsia, distortion of outlines, central photophobia	Calcarine cortex.
9	Complex hallucinations	Non dominant hemisphere.

POSTERIOR CEREBRAL CIRCULATION SYNDROME

Central territory

no	Signs and symptoms	Structures involved
1	Thalamic syndrome: sensory loss (all modalities), spontaneous pain and dysesthesias, choreoathetosis, intention tremor, spasms of hand, mild hemi paresis	Postero ventral nucleus of thalamus; involvement of the adjacent sub thalamus body or its afferent tracts
2	Thalamo perforate syndrome: crossed cerebellar ataxia with ipsilateral third nerve palsy (Claude's syndrome)	Dentatothalamic tract and issuing third nerve
3	Weber's syndrome: third nerve palsy and contra lateral hemiplegia	Third nerve and cerebral peduncle
4	Contra lateral hemiplegia	Cerebral peduncle
5	Paralysis or paresis of vertical eye movement, skew deviation, sluggish pupillary responses to light, slight miosis and ptosis (retraction nystagmus and "tucking" of the eyelids may be associated)	Supranuclear fibers to third nerve, interstitial nucleus of Cajal, nucleus of Darkschewitsch, and posterior commissure
6	Contra lateral rhythmic, ataxic action tremor; rhythmic postural or "holding" tremor (rubral tremor)	Dentatothalamic tract

DIAGNOSTIC EVALUATION OF ISCHAEMIC STROKE [17]

The diagnostic evaluation should include parallel assessment of the following.

1. Imaging of the infarct
2. Vascular studies
3. Cardiac evaluation
4. Hematological and other blood testing

Imaging

CT scan brain is done to differentiate hemorrhage from infarction. Investigations must be ordered based on the possible etiology. Cerebral arteriography is needed if intra arterial thrombolysis is contemplated. Angiogram is done if vascular stenosis is suspected and if it is negative or if posterior circulation is suspected then ,Tran cranial Doppler is done.

CT versus MRI

1. Only a minority infarction demonstrated within 24 hours on CT. MRI documents infarct as early 6 hours
2. Anatomic extent and vascular distribution are better delineated in MRI. Small infarctions are easily seen and white matter better visualized.
3. Posterior fossa infarctions are better visualized in MRI.

CT scan changes in cerebral infarction [18]

Hyper acute infarct < 12 hours

- normal (50 – 60%)

- Hyper dense artery(25 – 50%)
- Obscuration of Lentiform nuclei

Acute infarct 12 to 24hours

- Low density basal ganglia
- Loss of grey white interfaces
- Sulcal effacement

1 to 3days

- Increasing mass effect
- Wedge shaped low density area that involves both grey and White matter
- Hemorrhagic transformation may occur(basal ganglia and cortex are common sites)

4 to 7days

- Gyral enhancement
- mass effect, edema persist

1 to 8 weeks

- Contrast enhancement persists
- mass effect resolves

months to years

- encephalomalacic changes

- volume loss
- calcification is rare

CLINICAL CORRELATION OF HYPERGLYCEMIA AND INFARCT PROGRESSION

Although experimental studies have clarified several mechanisms by which hyperglycemia influences the destiny of ischemic brain issue, studies bridging the gap between clinical stroke and experimental models have been scare. Recent advances in Magnetic Resonance Imaging techniques have permitted Correlation of loss of penumbral tissue with elevated blood glucose, which was linked to increased brain lactate production[19]. Using a subcutaneous glucose sensor for continuous monitoring up to seventy two hours, the sane group could reproduce the findings that the infarcts expanded more in hyperglycemic patients and that hyperglycemia was independently associated with the infarct volume change[20]. This suggests that increased glucose not only reflects the initial volume of infracted tissue in the acute stage but is one of the true determinants of early infarct progression in men.

Neurological Pearls In Prognosis

factors predicting poor outcome[21]

1. age : more than 75 years
2. males : due to lack estrogen protective effect

3. risk factors : atrial fibrillation , DM , previous stroke
4. clinical findings
    - decreased consciousness at the onset
    - Presence of gaze deviation
    - Headache , nausea ,vomiting in first 24 hours
    - Elevated systolic BP >180 mm Hg on first day
    - Hyperthermia on admission
    - NIHSS score of 16 or more
    - Large vessel disease
5. Laboratory findings
    - High glutamine in plasma > 200 micromol / L
    - CRP concentration > 10.2 mg / l within 72 hours
    - Hyperglycemia > 7mmol / l
    - Platelet count < 150000 due to increased bleeding
6. Neuro imaging studies
    - Hyper density in a major intra cranial artery
    - Early CT changes within 6 hours of onset
    - >33 % of MCA territory involvement / multiple territory involvement with mass effect
    - Hemorrhagic transformation on follow up CT / on intra cranial Doppler persisting MCA occlusion for hours

- No flow on SPECT perfusion patterns
- Carotid artery occlusion on conventional angiogram
- MCA , basilar artery occlusion on angiogram
- MRI – abnormal PWI in diffusion and perfusion weighted imaging
- MRA – absence of MCA is associated with poor prognosis

PROGNOSIS AND HYPERGLYCEMIA

Already ample literature has demonstrated that hyperglycemia on admission is associated with worsened clinical outcome as reviewed in a systemic overview of thirty three studies[22]. Glycemic control may be indicated also in non diabetic patients, in which stress hyperglycemia was associated with a three fold risk of fatal thirty day outcome and 1.4 fold risk in of poor functional outcome. Good glycemic control seems warranted also in hemorrhagic stroke[15], although More clinical information is needed in this area. At least two clinical trials have Recently been initiated to examine the efficacy of early insulin therapy in acute stroke[20,23]. Still there is no evidence to prove that the reversal of hyperglycemia improves the prognosis, as it has been demonstrated to do in acute myocardial infarction[24]and in critically ill post surgical patients[25].

TREATMENT

The first goal is to prevent or reverse brain injury

The second goal is to obtain an accurate understanding of the stroke mechanism so one can halt progression of brain injury or begin to prevent a second stroke

Treatments designed to reverse or lessen the amount of tissue infarction fall Within five categories:

(1) Medical support

(2) Thrombolysis

(3) Anticoagulation

(4) Antiplatelet agents

(5) Neuroprotection

(1)Medical Support

1.When cerebral infarction occurs, the immediate goal is to optimize cerebral perfusion in the surrounding ischemic area

2 .Preventing the common complications of bedridden patients like infections (pneumonia, urinary tract, and skin), deep venous thrombosis with pulmonary embolism.

3.Elevated BP should not be lowered unless there is malignant hypertension or concomitant myocardial ischemia. If the blood pressure is low, raising it is advisable, using intravenous fluids or vasopressor drugs to enhance perfusion within the ischemic penumbra.

4. Treatment of cerebral edema if necessary

(2)Thrombolysis

The use of thrombolytic agents in acute cerebral infarction has been studied extensively. Angiography performed within a few hours of infarction frequently demonstrates arterial occlusions corresponding to patients presenting signs and symptoms. It is this association of arterial occlusion with acute neurological symptoms that prompted the study of thrombolytic agents in stroke patients.

Agent used for this purpose is intravenous recombinant tissue plasminogen activator (rtPA)

Indication [26]

Clinical diagnosis of ischemic stroke with clear symptom onset within 3 hours

CT scan showing no hemorrhage or significant edema

Age more than 18 years, Consent by patient or surrogate

Contraindication [26]

Sustained BP > 185/110

Platelets < 100,000; HCT< 25%; glucose < 50 or > 400

Use of heparin within 48 h and prolonged PTT, or elevated INR

Rapidly improving symptoms

Prior stroke or head injury within 3 months; prior intracranial hemorrhage

Major surgery in preceding 14 days

Minor stroke symptoms

Gastrointestinal bleeding in preceding 21 days

Recent myocardial infarction, Coma or stupor

Administration of rtPA [27]

Intravenous access with two peripheral IV lines (avoid arterial or central placement)

Review eligibility for rtPA.

Administer 0.9 mg/kg intravenously (maximum 90 mg) IV as 10% of total dose by bolus, followed by remainder of total dose over 1 h.

Continuous blood pressure monitoring.

No other antithrombotic treatment for 24 h.

For decline in neurological status or uncontrolled blood pressure, stop infusion, give cryoprecipitate, and reimage brain emergently

Avoid urethral catheterization for > 2 h.

(3)Anticoagulation

The role of anticoagulation in atherothrombotic cerebral ischemia is uncertain. Heparinization is generally accomplished by beginning an infusion without bolus and is monitored to maintain the activated PTT at approximately twice normal.

This regimen is maintained for 2 to 5 days. During this time the patient is monitored for hemorrhagic complications, the evaluation is completed

decision is made regarding the need for carotid endarterectomy, long-term anticoagulation, or an antiplatelet therapy.

If long-term anticoagulation is chosen, warfarin is administered and heparin discontinued when the international normalized ratio (INR) is in the range of 2 to 3.

(4)Antiplatelet Agents

Aspirin is the only antiplatelet agent that has been prospectively studied for the treatment of acute ischemic stroke. The use of aspirin within 48 h of stroke onset reduced both stroke recurrence risk and mortality minimally

(5)Neuroprotection

Neuroprotection is the concept of providing a treatment that prolongs the brain's tolerance to ischemia long enough to allow other measures to be employed to mitigate ischemia.

Hyperthermia , Hyperglycemia are the agents which increases the size of the infarct, hence hypothermia and normalizing blood sugar are powerful neuroprotective agents.

HYPERGLYCEMIA AND THROMBOLYTIC THERAPHY OF ACUTE ISCHEMIC STROKE

In several thrombolysis trials, hyperglycemia has been associated With hemorrhagic events [28] and was reconfirmed in recently[29] as well as in a re-Analysis of the NINDS rt-PA trial[30]. In the latter study, as increasing admission

glucose level was independently associated with decreased odds for neurological improvement (odds ratio, OR = 0.76 per 100 mg/dl increase in admission glucose) and the odds ratio for symptomatic intra cerebral hemorrhagic was 1.75 per 100 mg/dl increase in admission glucose (95 % CI 1.11 to 2.78, p = 0.02). The relationship was weaker after excluding patients with intra cerebral hemorrhage, suggesting admission hyperglycemia may exert its hazard in part through hemorrhagic events. However another recent study by Alvarez-Sabin et al found admission glucose more than 140 mg/dl (OR 8.4, CI 1.8 to 40.0) to be the sole independent predictor of poor functional outcome at three months in patients with recanalization within six hours, even after excluding the patients symptomatic intra cerebral hemorrhage[31]. The same was not true for the patients who did not recanalize, which leads to speculation that might partially preclude the beneficial effect of r-tPA and early reperfusion.

PRIMARY AND SECONDARY PREVENTION

Atherosclerosis Risk Factors

Hypertension is the most significant of the risk factors; in general, all hypertensives should be treated. Coronary artery disease is the most common cause of death in patients with cerebrovascular disease, treatment of hypercholesterolemia seems prudent for both the heart and brain. Tobacco

smoking should be discouraged in all patients

Antiplatelet Agents

Platelet antiaggregation agents can prevent atherothrombotic events, including TIA and stroke, by inhibiting the formation of intra-arterial platelet aggregates. These can form on diseased arteries, induce thrombus formation, and occlude the artery or embolize into the distal circulation.

The antiplatelet agents used most for this purpose are :

Aspirin,

Clopidogrel, and

The combination of aspirin plus extended-release Dipyridamole

Anticoagulation Therapy

ATHEROTHROMBOTIC STROKE

There are few data to support the use of long-term warfarin for preventing atherothrombotic stroke, either intra cranially or extra cranially.

EMBOLIC STROKE

Several recent trials demonstrated that anticoagulation (INR range 2 to 3) in patients with chronic non valvular (non rheumatic) atrial fibrillation prevents cerebral embolism and is safe.

For primary prevention and for patients who have experienced

stroke or TIA, anticoagulation with warfarin reduces the risk by about 65% and clearly outweighs the 1% per year rate of major bleeding complication.

Because of the high annual stroke risk in untreated rheumatic heart disease, primary prophylaxis against stroke has not been studied in a double-blind fashion. These patients generally receive long-term anticoagulation.

Anticoagulation also reduces the risk of embolism in acute myocardial infarction. Most clinicians recommend a 3-month course of anticoagulation when there is anterior Q-wave infarction, substantial Left ventricular dysfunction ,congestive heart failure, mural thrombosis, or atrial fibrillation .Warfarin is recommended long-term if atrial fibrillation persists. Thromboembolism is one of the most serious complications of prosthetic heart valve implantation. Anticoagulation has been proven effective for preventing strokes in this situation, while antiplatelet therapy alone has not.

However, coupled with warfarin anticoagulation, aspirin adds substantial benefit. A greater degree of anticoagulation (INR of 3 to 4, depending on valve type) is recommended for prosthetic heart valve patients.

SURGICAL THERAPY

Surgery for atherosclerotic occlusive disease is largely limited to carotid endarterectomy for plaques located at the origin of the internal carotid artery in the neck

Carotid endarterectomy is a proven effective prophylaxis against stroke and TIA.

Stroke Centers and Rehabilitation

Comprehensive stroke units that care for the acute patient followed by rehabilitation services have been shown to improve neurological outcomes and reduce mortality.

Proper rehabilitation of the stroke patient includes early physical, occupational, and speech therapy. It is directed toward educating the patient and family about the patient's neurological deficit, preventing the complications of immobility (e.g., pneumonia, deep vein thrombosis and pulmonary embolism , pressure sores of the skin, muscle contractures), and providing encouragement and instruction in overcoming the deficit. The goal of rehabilitation is to return the patient to home and to maximize recovery by providing a safe, progressive regimen suited to the individual patient

INTRA CEREBRAL HEMORRHAGE

Intra cerebral hemorrhage accounts for approximately 15%-25% of the strokes. The overall mortality for this type subtype of stroke is from 25 % to 60% In nearly 70% of patients Hypertension is the commonest cause of bleed.

The lipohyalinosis of the small intraparenchymal arteries is the leading cause of hemorrhage.

. The micro aneurysms of charcot and Bouchard is uncertain, but they are found at anatomical sites preferentially affected by ICH.

Hyperglycemia And Hemorrhagic Stroke :

Incidence of hemorrhagic stroke is higher in diabetes either known diabetes or newly detected diabetes. This is due to the fact that blood brain barrier is disrupted by chronically elevated hyperglycemia which is dealt earlier in the topic 'thrombolysis of ischemic patients with hyperglycemia'

The non Hypertensive causes include following :

1. bleeding disorders, anticoagulant and fibrinolytic treatment
2. cerebral amyloid angiopathy
3. granulomatous angitis of the CNS
4. sympathomimetic agents
5. trauma
6. hemorrhagic infarction
7. vascular malformations
8. Intra cranial tumors

clinical features:

symptoms of increased ICT

symptoms that are specific for the location of the hematoma

focal neurological deficits

Imaging

The CT scan is sensitive to the high density fresh blood in the parenchyma while MRI can determine the time duration between the hemorrhage and the MRI examination

common sites :

1 . Putamen - 35 %

2 . Lobar – 25 %

3 . Thalamus – 10-15 %

4 . Caudate nucleus – 5 %

5 . Pons – 5 %

6. Cerebellum – 5 – 10 %

Treatment

Control of hypertension

Air way maintenance if the GCS is less than 8

Treatment of coagulation abnormalities

Protamine sulfate is used if the hemorrhage is due to heparin Cryoprecipitate is used if the hemorrhage is due to heparin

Measures for the prevention of further elevation of ICP

Routine anticonvulsants not recommended in patients who do not have seizures at onset due to negligible risk of subsequent epilepsy in them.

Patients with lobar hemorrhage and cerebellar hemorrhage can be managed surgically whereas the deep hemorrhages are managed medically.

MORTALITY IN STROKE :

Early mortality (first thirty days) :

Generally death occurs with in first 1 days and is secondary to large infarction leading to cerebral edema and raised Intra cranial tension. These patients can be clinically identified by

1. Severe fall in level of consciousness.
2. Gaze paresis.
3. Hemianopia.
4. Dilated pupil on the infracted side.
5. Abnormal type of respiration.

In such patients CT scan will reveal large hemispherical infarct with edema and midline shift and or brain stem infarct.

Other causes after 10 days :

1. Aspiration pneumonitis.
2. Bed sores.

3. Infections.
4. Pulmonary embolism.

Late mortality :

Important causes are IHD and recurrent stroke.

SUMMARY OF EVIDENCE SUPPORTING A DETRIMENTAL ROLE FOR ELEVATED GLUCOSE IN STROKE

1. Experimental ischemic damage is worsened by hyperglycemia
2. Experimental ischemic damage is reduced by glucose reduction
3. Early hyperglycemia is associated with clinical infarct progression in brain imaging
4. Early hyperglycemia is associated with hemorrhagic conversion in stroke
5. Early hyperglycemia is associated with poor clinical outcome
6. Early hyperglycemia may reduce the benefits of recanalization
7. Immediate insulin therapy reported beneficial in acute myocardial infarction and surgical critical illness.

CONCLUSION

This recent evidence supports that acutely elevated, predominantly stress related hyperglycemia is associated with poor outcome

such as dependent state or intra cerebral hemorrhage. Through different biochemical mechanisms , increased glucose in setting of cerebro vascular insults probably accelerates the course of ischemic injury, also in the boundary regions with milder perfusion deficit. Although admission hyperglycemia has been already demonstrated to be risk factor for symptomatic hemorrhage and worsened outcome after thrombotic therapy, there is perhaps not enough evidence to withhold thrombolysis from hyperglycemic patients within the three hour time window. How ever restoration of normoglycemia as soon as possible should be encouraged ,although conclusive evidence of decreased risk with this approach is lacking. Especially the non diabetic patients may be a risk of further brain damage if hyperglycemia prevails . The recent evidence summarized above urges corroboration in randomized controlled trial of the efficacy of immediate sugar control, and determination of where the level of target glucose concentration of relatively different current values in the published guidelines (EUSI < 10 mmol/l; ASA < 300 mg/dl) [7] should be set. In the interim, we should fare well with adhering to good general stroke management, including control of blood glucose , normalization of body temperature, fluid balance and homodynamics or we may otherwise risk the favorable outcome even in the patients with early recanalization.

MATERIALS AND METHODS

4.MATERIALS AND METHODS

A total of hundred and nine patients of acute stroke admitted in the department of medicine, Government Stanley Hospital, Chennai between August 2005 to May 2006 were studied. The Patients were selected on the following basis

INCLUSION CRITERIA:

1. Patients should be above the age of forty
2. Patients should have been admitted within twenty four hours of onset of symptoms
3. This should be the first cerebro vascular accident for the patient
4. Blood sugar recorded with in twenty four hours of the onset of stroke

EXCLUSION CRITERIA:

1. Patients admitted after twenty four hours of stroke
2. Those patients who received intravenous glucose before or during study period
3. Patients with reliable information about diabetes could not be obtained
4. Patients who died before it could be established whether or not they had diabetes
5. Illness presented with stroke like symptoms

Out of the hundred and nine patients, nine were dropped as follow up could not be done.

Complete history was taken, clinical examination was done and clinical diagnosis for each patient was arrived.

Blood pressure measurement , blood sugar , urea , creatinine , electrolytes, hemoglobin, total count, differential count; urine sugar, albumin, deposits; electrocardiogram and chest X ray done for all patients

The severity of stroke for each patient is calculated based on NIH stroke scale, NIHSS[32] which takes the following clinical findings in to account and each criteria awarded specific points

1a Level of conscious

Alert	0
Drowsy	1
Stuporous	2
Comatose	3

1b LOC questions

Answers both correctly	0
Answers one correctly	1
Incorrect	2

1c LOC commands

Obeys both correctly	0

Obeys one correctly	1
Incorrect	2
2 Best gaze	
Normal	0
Partial gaze palsy	1
Forced deviation	2
3 Visual	
No visual loss	0
Partial hemianopia	1
Complete hemianopia	2
Bilateral hemianopia	3
4 Facial palsy	
Normal symmetric	0
Minor paralysis	1
Partial paralysis	2
Complete paralysis	3
5 Best motor arm/ leg (right/left)	
No drift	0
Drift	1
Some antigravity effect	2
No antigravity effect	3

No movement 4

6 Limb ataxia

Absent 0

Present in one limb 1

Present in both limbs 2

7 Sensory

No sensory loss 0

Mild to moderate sensory loss 1

Total sensory loss 2

8 Best language

Normal, no aphasia 0

Mild to moderate aphasia 1

Severe aphasia 2

Mute, global aphasia 3

9 Dysarthria

Normal 0

Mild to moderate 1

Severe 2

10 Extinction/ inattention

No abnormality 0

Visual/ tactile/ spatial/ personal inattention 1

The points were added, with a maximum of thirty points.

Once clinical diagnosis of acute stroke is made venous blood sample is taken, with in twenty four hours of onset of symptoms, and sent to laboratory for glucose estimation.

In patients with blood sugar more than 6.1 mmol/l(110 mg/dl) [22] and with out a history of diabetes , Hemoglobin A 1c was performed. (Hemoglobin A1c is structurally similar to hemoglobin A except for the addition of glucose Group to the terminal amino acid of the beta chain of the hemoglobin Molecule (glycosylation). Therefore hemoglobin A 1c is a function of the exposure of the red blood cells to glucose. Since the glucose linkage to hemoglobin is relatively stable, Hemoglobin A1c accumulates through out of the life span of erythrocyte and its concentration reflects the integrated blood glucose concentration over a period approximating to the half life of erythrocytes i.e. six to eight weeks. Therefore measurement of hemoglobin A 1c helps to monitor the overall degree of diabetic control achieved). The normal range of Hemoglobin A 1c is 3.8% to 6.4%[33]. Hence the patients can be classified into four groups

Blood sugar less than 6.1 mmol/l : **Non diabetic (euglycemic)**

History of diabetes : **Known diabetics**

Blood sugar more than 6.1mmol/l, no history of diabetes, and hemoglobin A1c

more than 6.4% : **Newly detected diabetics**

Blood sugar more than 6.1 mmol/l, no history of diabetes, and hemoglobin A1c less than 6.4% : **Stress hyperglycemics**

Then computerized tomography, CT, of the brain was performed in all patients to :

Confirm the diagnosis

Detect the type of stroke

Detect the size of lesion (small < 5mm; Medium 5 – 10 mm; Large > 10 mm or involving more than one vascular territory)

Locate the site of lesion

Identify the presence of cerebral edema or midline shift

The patients were followed up for thirty days and outcome in the form of death ; poor, moderate and good improvement were recorded. Patients who were unable to return to any form of work[34], persistent disability[35], need for residential placement[36],dependent in activities of daily living[37], and stable deficit with no recovery[38] were classified as those with poor outcome. Patient whose symptoms improved, who were independent in attending day to day activities, improvement in motor function and aphasia and no persistent disability were grouped as patients with good outcome. Patients who fared in between these two groups were grouped as those with moderate outcome.

OBSERVATION AND RESULTS

5. OBSERVATION AND RESULTS

SEX DISTRIBUTION :

Sex	No of cases	%
male	66	66%
female	34	34%

AGE WISE DISTRIBUTION :

Age	Male	Female	Total	%
41 - 50	16	4	20	20
51 - 60	28	10	38	38
61 - 70	9	11	20	20
71 - 80	10	9	19	19
>80	3	0	3	3
	66	34	100	100

RISK FACTORS :

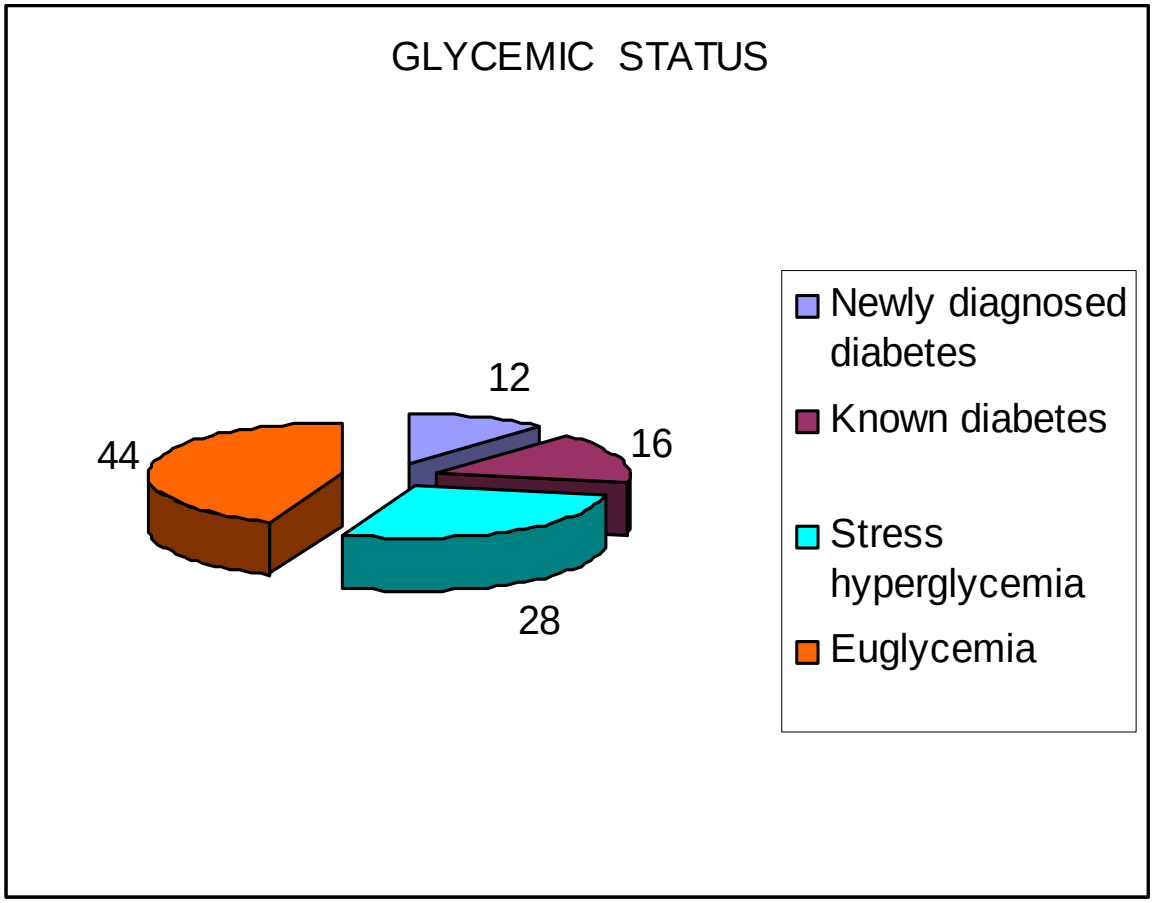
Risk factors	Male	%	Female	%	Total
hypertension	42	63.64	24	36.36	66
diabetes	17	60.71	11	39.29	28
Hyper cholesterolemia	10	71.43	4	28.56	14
Atrial fibrillation	0	0	1	100	1
Coronary artery disease	4	66.66	2	33.33	6
smoking	40	100	0	0	40
alcohol	25	96.15	1	3.85	26

CLINICAL PRESENTATION :

Clinical presentation	Male	%	Female	%	Total
Right hemiplegia	38	69.1	17	30.9	55
Left hemiplegia	25	64.1	14	35.9	39
Faciobrachial monoplegia	2	40	3	60	5
Cerebellar symptoms	1	100	0	0	1
Loss of consciousness	31	60.8	20	39.2	51
Hemianopia	2	66.6	1	33.3	3
Aphasia	22	57.9	16	42.1	38
Bladder and Bowel involvement	16	59.3	11	40.7	27

GLYCEMIC STATUS :

Glycemic status	Total
Euglycemia	44
Stress Hyperglycemia	28
Known Diabetes	16
Newly diagnosed Diabetes	12

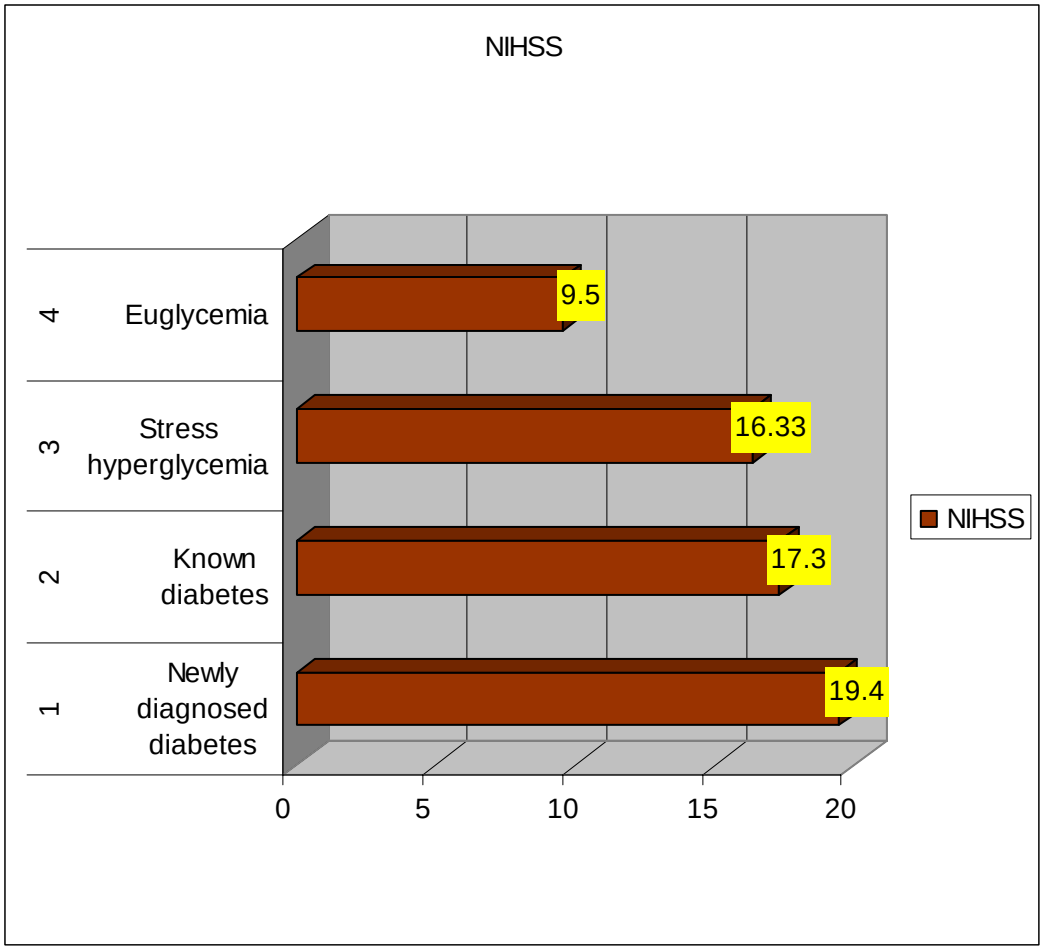

GLYCEMIC STATUS
12
16
28
44
Newly diagnosed diabetes
Known diabetes
Stress hyperglycemia
Euglycemia

STROKE SEVERITY :

Glycemic status	NIHSS
Euglycemia	9.5
Stress hyperglycemia	16.33
Known diabetes	17.3
Newly diagnosed diabetes	19.4

Statistical Analysis :

	N	Mean	Std. Deviation	Oneway ANOVA F_test
Euglycemia	44	9.50	6.760	
Stress hyperglycemia	28	16.32	7.040	
Known diabetes	16	17.38	6.561	**F=11.85 P=0.001**
Newly detected diabetes	12	19.42	5.248	
Total	100	13.86	7.671	

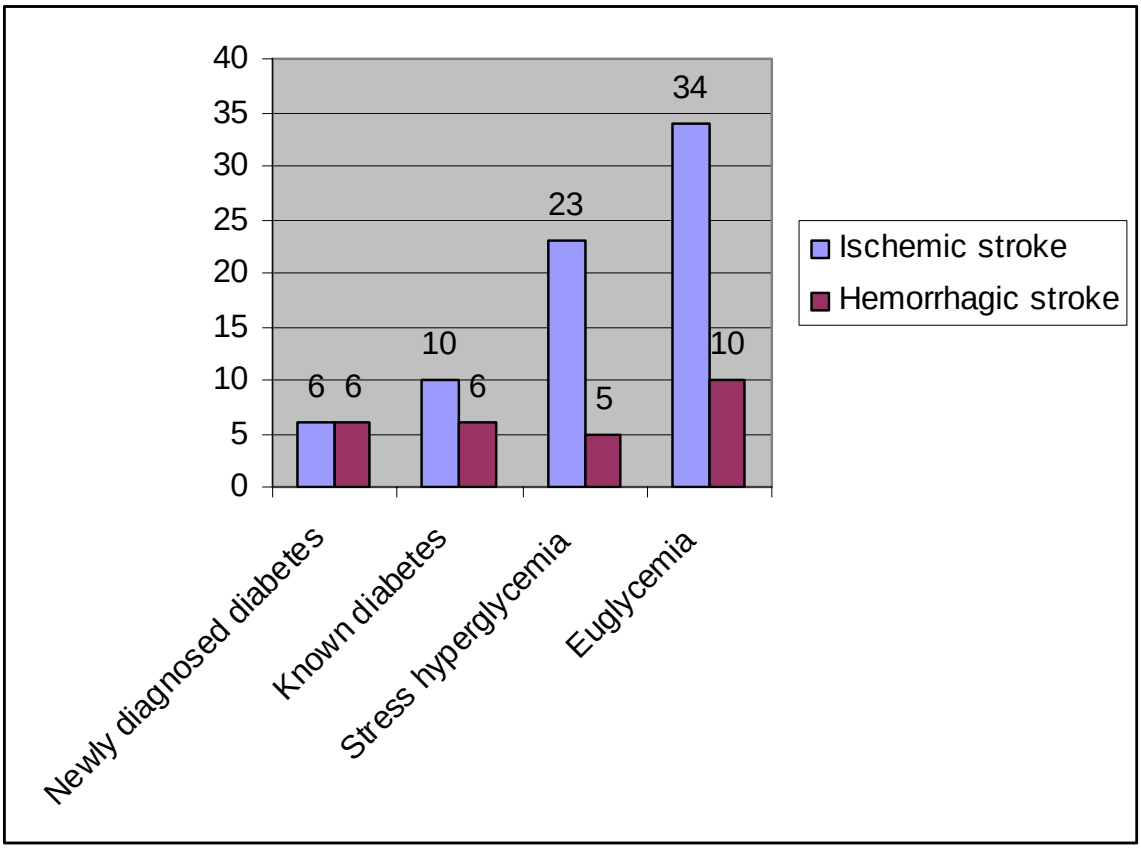

NIHSS
4
Euglycemia
9.5
3
Stress hyperglycemia
16.33
2
Known diabetes
17.3
1
Newly diagnosed diabetes
19.4
0
5
10
15
20
NIHSS

Glycemic status	Ischemic stroke		Hemorrhagic stroke		Total
	NO	%	NO	%	
Euglycemia	34	77.27	10	22.73	44
Stress hyperglycemia	23	82.14	5	17.86	28
Known diabetes	10	62.5	6	37.5	16
Newly diagnosed Diabetes	6	50	6	50	12

SIZE OF THE LESION :

Glycemic status	Total	Small	Medium	Large
Euglycemia	44	29	8	7
Stress Hyperglycemia	28	1	14	13
Known Diabetes	16	2	6	8
Newly Detected Diabetes	12	0	6	6

$\chi^2 = 42.5$, $p = 0.001$.


40
35
30
25
20
15
10
5
0
6
6
10
6
23
5
34
10
Ischemic stroke
Hemorrhagic stroke
Newly diagnosed diabetes
Known diabetes
Stress hyperglycemia
Euglycemia

CLINICAL OUTCOME :

Glycemic Status	Total	Death		Poor		Moderate		Good	
		NO	%	NO	%	NO	%	NO	%
Euglycemia	44	7	15.91	2	4.54	6	13.64	29	65.91
Stress Hyperglycemia	28	10	35.71	8	28.57	9	32.14	1	3.54
Known Diabetes	16	7	43.75	3	18.75	5	31.25	1	6.25
Newly diagnosed diabetes	12	6	50	4	33.33	2	16.67	0	0

Statistical Analysis of Out Come :

		outcome				Total
		Good	Moderate	Poor	Death	
group	**Euglycemia**	29	6	2	7	44
	Stress hyperglycemia	1	9	8	10	28
	Known diabetes	1	5	3	7	16
	Newly detected diabetes	0	2	4	6	12
Total		31	22	17	30	100

$\chi 2=48.3$, P=0.001

OUTCOME IN STROKE SUBTYPES :

GLYCEMIC STATUS			outcome				Total
			Good	Moderate	Poor	Death	
HEMORRHAGE	group	Euglycemia	3	3	0	4	10
		Stress hyperglycemia	0	1	1	3	5
		Known diabetes	0	3	2	1	6
		Newly detected diabetes	0	2	3	1	6
	Total		3	9	6	9	27
INFARCT	group	Euglycemia	26	3	2	3	34
		Stress hyperglycemia	1	8	7	7	23
		Known diabetes	1	2	1	6	10
		Newly detected diabetes	0	0	1	5	6
	Total		28	13	11	21	73

Hemorrhage: $\chi 2=12.75$, P=0.17 **(Not significant)**

Infarct : $\chi 2=50.6$, P=0.001 **(Significant)**

OUTCOME OF STROKE IN NON DIABETES PATIENTS :

		Ischemic stroke		Hemorrhagic stroke	
		Euglycemia	Stress Hyperglyc-emia	Euglycemia	Stress Hyperglyce-mia
Total		34	23	10	5
NIHSS		7.62	15.56	14.4	19.8
Death	No	3	7	4	3
	%	8.82	30.43	40	60
Poor	No	2	7	0	1
	%	5.58	30.43	0	20
Moderate	No	3	8	3	1
	%	8.82	34.78	30	20
Good	No	26	1	3	0
	%	76.47	4.35	30	0
Average blood Glucose level		91.68	144.43	102.12	240.6

CORRELATION OF SUGAR LEVEL AND OUTCOME IN BOTH SUBTYPES OF STROKE:

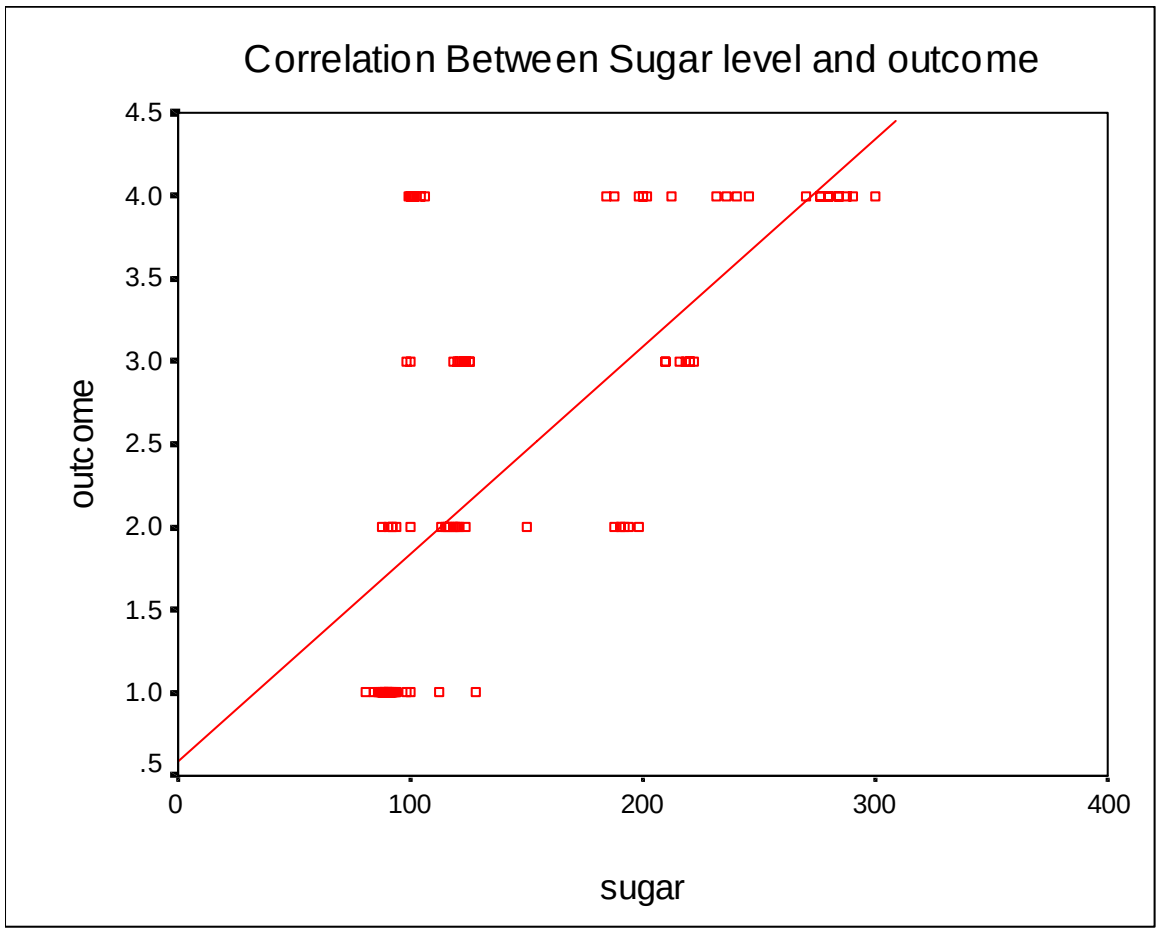
			Outcome				Total
			Good	Moderate	Poor	Death	
HEMORRHAGE	SUGAR	A	3	3	0	4	10
		B	0	1	1	0	2
		C	0	5	0	0	5
		D	0	0	5	5	10
	Total		3	9	6	9	27
INFARCT	SUGAR	A	26	3	2	3	34
		B	1	8	7	0	16
		C	1	2	0	3	6
		D	0	0	2	15	17
	Total		28	13	11	21	73

A <110 mg/dl ; **B** = 110-125 mg/dl ; **C** = 126-199 mg/dl ; **D** > 199 mg/dl

Hemorrhage : $\chi 2=26.1$, P=0.001 (Significant)

Infarction : $\chi 2=81.9$, P=0.001 (Significant)

Positive correlation, r = 0.71, p = 0.001.


Correlation Between Sugar level and outcome
outcome
4.5
4.0
3.5
3.0
2.5
2.0
1.5
1.0
.5
0
100
200
300
400
sugar

DISCUSSION

6.DISCUSSION

AGE, SEX AND RISK FACTORS:

In our study of hundred patients majority of them belonged to male sex showing a male preponderance which is commonly seen in most studies. Majority of the patients, thirty eight, were between the age group of 51 to 60. Among the hundred patients 66 had hypertension, 28 had diabetes, 14 had hypercholesterolemia, 6 had previous history of myocardial infarction, and one lady had atrial fibrillation. More than half of the male patients were smokers and one third had history of alcohol intake. Sixty patients had right sided weakness and forty patients had left sided weakness.

GLYCEMIC STATUS :

Among the hundred patients in our study group, 56 patients had elevated admission day blood glucose level and 44 patients had normal blood glucose values. Diabetes was noticed in 28 patients and stress hyperglycemia in another 28 patients . In ischemic stroke group stress hyperglycemia amounted to one third of the patients and one fifth in hemorrhagic group.

SEVERITY OF STROKE :

Severity of stroke was assessed with NIH Stroke scaling system. Admission day hyperglycemic patients had a higher score when compared to Euglycemic patients (17.27 vs. 9.5 respectively) , which was statistically

significant with p = 0.001. Among the admission day hyperglycemic patients newly detected diabetes had the highest mean NIHSS. Hence an elevated blood sugar at the time of stroke resulted in severe stroke.

SIZE OF STROKE:

The size of the lesion were analyzed with the help of CT scan brain. Most of the euglycemic patients had small sized infarcts and hemorrhage whereas majority of the admission day hyperglycemic patients had large sized lesion with edema and midline shift. These data's were statistically significant with p = 0.001. Hyperglycemia by virtue of increased anaerobic metabolism, increased brain lactate, impaired mitochondrial function, vascular disease, increased free radical production, increased expression of c-fos and cox-2 causes severe brain injury and large sized infarcts. Hyperglycemia can disrupt the blood brain barrier resulting in large hemorrhage and hemorrhagic transformation of infarcts.

TYPE OF STROKE :

Among the euglycemic group three fourth of the patients had ischemic stroke and one fourth had hemorrhagic stroke. Among patients with admission day hyperglycemia one third of the patients had hemorrhagic stroke. In the newly detected diabetes patients half of them had hemorrhagic stroke. Our study shows an increasing incidence of hemorrhagic stroke among

diabetes patients.

OUTCOME OF STROKE :

In this study of hundred acute stroke patients, euglycemic patients had a better outcome when compared to admission day hyperglycemic patients.

Euglycemic patients had a better recovery after acute stroke. Sixty five percent of euglycemic patients had a good functional recovery. On the contrary only three percent of admission day hyperglycemic patients had good functional recovery at the end of thirty day follow up.

Early inpatient mortality was high in admission day hyperglycemic patients. Forty percent of the admission day hyperglycemic patients died with in the first thirty days. In the euglycemic patients the early case fatality rate was only fifteen percent. Hence there was a two and a half fold increased risk of early mortality in admission day hyperglycemic patients when compared to euglycemics. Poor outcome was noticed in twenty seven percent of admission day hyperglycemic patients and in four percent of euglycemic patients.

This study of hundred acute stroke patients shows that admission day elevated blood glucose level was associated with a high early mortality rate and an increased risk of poor functional recovery.

These data's were statistically significant with $\chi 2=48.3$ and $p=0.001$.

In the ischemic stroke group early mortality rate was 8.82 % in euglycemic patients and 46.15 % in hyperglycemic patients. Poor outcome was noticed in 5.88 % in euglycemics and 23.3 % in hyperglycemics. Hence hyperglycemia was associated with an increased early mortality rate and poor functional outcome in ischemic stroke group which was also statistically significant with : $\chi 2=50.6$ $P=0.001$.

In the hemorrhagic patients the early mortality in hyperglycemic patients was 29.4 % and 40 % in euglycemic patients which was statistically insignificant with $\chi 2=12.75$ $P=0.17$.

In the non diabetes ischemic stroke patients stress hyperglycemia had worst outcome when compared to euglycemic group. The early mortality rate was 30.43 % in stress hyperglycemics and 8.82 % in euglycemics. Hence this study shows a three and a half fold increased risk of mortality in non diabetes stress hyperglycemic patient when compared to non diabetic euglycemic patients which was also statistically significant with p=0.001. However similar significance was not noticed in the hemorrhagic group.

Our study clearly shows a positive correlation (r = 0.71, p = 0.01) between admission day sugar value and the outcome of stroke. Higher admission day elevated blood glucose level has increased mortality and high risk of poor functional recovery.

COMPARISION WITH OTHER STUDIES :

According to Perttu J. Lindsberg and Risto o Roine[39] hyperglycemia was noted in two third (66%) of all ischemic stroke patients. In our study hyperglycemia was noticed in 56% of patients in general and in 55% of patients with ischemic stroke. In their study known diabetes and newly diagnosed diabetes contributed one third of cases (33%). In our study the same group contributed to 28%.

A study published in European journal of Neurology, 2002[40] concluded that elevated glucose level after acute stroke is associated with higher stroke severity than those with normal level. The mean NIHSS was 9.5 in euglycemics and 17.27 in hyperglycemic patients in our study.

In the journal of clinical endocrinology and metabolism, 2002[41] a study confirmed that patients with newly detected hyperglycemia had a significant higher early mortality and a lower functional outcome than patients with a history of diabetes or normoglycemia. Our study in hundred acute stroke patients had the same results.

Sarah E capes et al[22] analyzed thirty two similar studies and concluded that hyperglycemic patients had threefold increased early mortality than euglycemic patients. After ischemic stroke admission hyperglycemia was associated with three fold increased 30 day mortality than euglcemics[22]. After hemorrhagic stroke, admission hyperglycemia was not associated with higher

mortality in either diabetic or non diabetic patients[22]. In our study, in ischemic patients, who had elevated admission day glucose level experienced a three and a half fold increased early mortality than euglycemics. Hemorrhagic patients with admission hyperglycemia did not show a statistically significant early mortality when compared to their euglycemic counter part. Similar results were noticed in non diabetic patients. Non diabetic stress hyperglycemic patients with ischemic stroke had three and a half fold increased early mortality when compared to euglycemics. In the diabetic group since the sugar value before the onset of stroke was not known, the effect of stress in diabetic group could not be studied.

The study clearly shows an increased early mortality rate and poor functional recovery in patients with diabetes and stress hyperglycemia when compared to euglycemics. Hence there is an urgent need to confirm the improvement in these patients by normalizing blood sugar. Several trails are now under way to improve the out come of Stroke by normalizing the blood glucose with human recombinant insulin. Stephan M. Vinychuk et[42] al showed that administration of insulin to patients with hyperglycemia improves functional recovery and vital activity of mild to moderate ischemic stroke patients. However , other clinical benefits of the insulin therapy remain to be determined.

CONCLUSION

8.CONCLUSION

There is a linear correlation between admission day hyperglycemia and stroke in its severity, size and outcome . The combined diabetes and stress hyperglycemics are found to have larger sized severe stroke and poor functional outcome in the form of increased mortality. There is a good correlation between admission day glucose level and the outcome in ischemic stroke. Admission day elevated glucose level was a significant predictor of mortality and poor functional outcome after acute stroke. Hence, restoration of normoglycemia as soon as possible should be encouraged though conclusive evidences are lacking. In the interim, we should fare well with adhering to good general stroke management, normalization of body temperature, fluid balance and hemodynamics or we may otherwise risk the favorable outcome even in the patients with normoglycemia.

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9.PROFORMA

NAME OF THE PATIENT :

AGE : **SEX :** **IP NO :**

ADDRESS : **OCCUPATION :**

CHIEF COMPLAINTS :

HISTORY OF PRESENTING COMPLAINTS :

PAST HISTORY :

HT / DM / TIA / PT / BA / COPD / CRF / CAHD / DCMP / RHD / AS / MVP / HEPATIC DISEASE / SEIZURES / PSYCHIATRIC DISORDERS

PERSONAL HISTORY : SMOKING, ALCOHOLISM, DIET

GENERAL EXAMINATION :

VITALS : BP, PULSE RATE, RESPIRATORY RATE AND PATTERN

CLINICAL EXAMINATION :

CENTRAL NERVOUS SYSTEM :

CARDIOVASCULAR SYSTEM :

RESPIRATORY SYSTEM :

ABDOMEN EXAMINATION :

SPINE AND CRANIUM :

CLINICAL DIAGNOSIS :

INVESTIGATIONS :

Blood sugar , urea , Serum creatinine

Serum electrolytes : sodium, Potassium

Blood hemoglobin , TC , DC, ESR

Urine albumin , sugar , deposits

Total cholesterol

Electrocardiogram

Chest X ray

CT scan brain

Glycosylated Hemoglobin when required

FINAL DIAGNOSIS :

PROGNOSIS (30 DAY FOLLOW UP) : Good / Moderate / Poor / Death

10.MASTER CHART

No	Name	Age	Sex	Past Hstory	BP	NIHss	SU	Hb A1c	CT BRAIN	prognosis
1	Salima Bee	80	F	Ht, Dm	170/100	15	190	-	L FP	Moderate
2	Padma	65	F	Ht	170/96	7	112	6.1	L F / VS	Good
3	Jaitun Bee	62	F	-	130/80	16	220	10.4	L PH	Poor
4	Ravi	48	M	Ht, S, A	170/100	8	92	-	R CH	Moderate
5	Munniammal	60	F	Dm, CAD	120/84	25	276	-	R FP / MLS	Death
6	Paramasivan	80	M	Ht	170/102	8	113	6	L PT / VS	Moderate
7	Anthonysamy	50	M	-	134/84	24	284	11	L PT / MLS	Death
8	Jambu	67	M	S, A, HCh	130/80	7	91	-	R F	Good
9	SAkunthala	70	F	Ht	170/100	7	81	-	L CG	Good
10	Naggapan	55	M	Dm, S	130/80	23	270	-	L FP / VS	Death
11	Munnusamy	48	M	Ht, S	170/96	23	101	-	L PT / VS	Death
12	Mohammed	75	M	Ht	164/90	4	90	-	L F	Good
13	Paputhai	75	F	-	130/80	10	116	6.1	R PT / VS	Moderate
14	Narasimman	60	M	Ht	190/110	25	184	6.1	R PT / MLS	Death
15	Chithirai	58	M	S, A	130/80	8	88	-	R F	Good
16	Sujatha	50	F	Ht, Dm, HCh	174/94	25	280	-	L FP / MLS	Death
17	Saleema Begam	75	F	Ht	200/110	26	198	6	R PT / MLS	Death
18	Narayanasamy	80	M	Ht	180/110	12	98	-	L F	Poor
19	Kuttan	50	M	-	130/84	16	216	10.2	L PH	Poor

No	NAME	Age	Sex	Past History	BP	NIHss	SU	Hb A1c	CT BRAIN	Prognosis
20	Panchacharam	53	M	Ht, S, A	210/110	23	188	6.1	R FP / MLS	Death
21	Manikandan	65	M	Ht, Dm	180/96	7	150	-	R CG	Moderate
22	Narayanan	66	M	Dm, DCMP	120/80	16	210	-	R PT / VS	Poor
23	Muniammal	60	F	Ht, A	180/110	7	92	-	L CH	Good
24	Santhanam	55	M	S, A	130/80	5	90	-	L CG	Good
25	Annamal	74	F	Ht	170/96	7	87	-	R CG	Good
26	Thasthagiri	80	M	-	120/80	8	115	6.1	L PT / VS	Moderate
27	Mani	55	M	Dm, S	130/80	24	276	-	R FP / MLS	Death
28	Boopalan	48	M	S, A, HCh	120/80	6	94	-	L CG	Moderate
29	Kaliyaperumal	49	M	Ht,S,A, CAD	130/84	5	90	-	L CEREBELAR	Good
30	Gajendran	53	M	Ht	190/110	25	232	6.1	L FP / MLS	Death
31	CHinnakulandhai	55	F	Ht	170/110	16	118	6.1	L PT / VS	Poor
32	Indrani	60	F	Dm, HCh	130/84	7	128	-	R CG	Good
33	Parvathy	55	F	-	120/80	10	108	-	R CG	Poor
34	Ranganayagi	65	F	Ht	200/110	25	246	6.1	R PH / MLS	Death
35	chinnathai	70	F	Ht	170/100	15	121	6.1	L PT / MLS	Poor
36	Rajendran	50	M	Ht, S	180/100	10	194	10.4	L PH	Moderate
37	Sonali	75	F	Ht	200/110	23	99	-	L PH / MLS	Death
38	Munusamy	60	M	-	130/80	4	89	-	L F	Good

No	NAME	Age	Sex	Past History	BP	NIHss	SU	Hb A1c	CT BRAIN	Prognosis
39	Ameer Bai	60	M	Ht, CAD	170/90	8	90	-	R CH	Moderate
40	Manohar	48	M	S, A	120/84	7	98	-	L CG	Good
41	Datchayanamoorty	60	M	Ht, Dm	180/96	23	280	-	R PT / MLS	Death
42	Arumugam	70	M	Ht	170/104	10	118	6.1	L PT / VS	Moderate
43	Palani	47	M	Dm, CAD	130/80	16	220	-	L FH	Poor
44	Abdul Kadhar	50	M	Ht, S, A	210/110	24	200	6.1	L FP / MLS	Death
45	Ramnaraynan	64	M	Ht	166/96	5	88	-	L F	Good
46	Leelavathy	55	F	-	200/110	25	236	6	R PH / MLS	Death
47	Vadivambal	70	F	Ht	170/110	16	210	10.6	L FPH	Poor
48	Irudhayaraj	48	M	Ht, Dm, S	180/90	16	218	-	R PH / MLS	Poor
49	Ponniammal	70	F	Ht, HCh	178/100	16	124	6	L FP / MLS	Poor
50	Vadivukarasi	75	F	Ht	170/100	24	290	10.2	L PT / MLS	Death
51	Baskar	47	M	Ht, S	174/96	25	300	11	R F / MLS	Death
52	Krishnan	55	M	Ht, S, A	176/96	5	84	-	L CH	Good
53	Muniandi	83	M	-	120/84	7	92	-	L CG	Good
54	Kanakavalli	75	F	Ht	200/110	25	212	6	R FP / MLS	Death
55	Ramalingam	55	M	S, A, HCh	120/80	9	120	6	R PT / VS	Moderate
56	Muniandi	52	M	Ht, S, A	210/110	26	102	-	L PH	Death
57	Prema	48	F	Hch	130/84	7	90	-	L F	Good

No	NAME	Age	Sex	Past History	10BP	NIHss	SU	Hb A1c	CT BRAIn	Prognosis
58	Mangaleswaran	75	M	-	130/80	7	89	-	R F	Good
59	Ulaganathan	83	M	Ht,Dm,S,MVP	174/94	23	280	-	L FP / VS	Death
60	Lakshmi	75	F	Ht, CAD	180/92	16	222	10.6	R PT / VS	Poor
61	Muniammal	47	F	Ht	180/110	6	89	-	L THA	Good
62	Lakshmi	53	F	Ht	180/102	7	90	-	L F	Good
63	Sundarambal	66	F	-	130/80	24	280	10.2	R PH / MLS	Death
64	Kuppan	66	M	Ht, S, A	180/110	7	90	-	L THA	Good
65	Gurusamy	60	M	Ht, S, HCh	174/100	23	288	11	L PT / MLS	Death
66	Nagaraj	53	M	S, A	120/80	10	121	6	R FP / VS	Moderate
67	Hawala	52	F	S, A	124/64	6	92	-	L CG	Moderate
68	Gowse moideen	80	M	Ht	200/110	18	125	6.1	R PH / MLS	Poor
69	Adhiammal	75	F	Ht, Dm	180/90	14	192	-	L FPH	Moderate
70	Thiruvenkadam	64	M	Ht, S, A	180/110	26	202	6	L PH / MLS	Death
71	Ranganayhan	56	M	Ht, S	170/110	6	94	-	R CG	Good
72	Ramalingam	63	M	Ht, S, A	164/90	9	119	6	L CG / VS	Moderate
73	Balaraman	49	M	Ht, Dm, S	190/96	24	280	-	L PH / MLS	Death
74	Chokkamal	55	F	Ht	170/80	5	90	-	R F	Good
75	Narmada devi	65	F	-	120/80	16	125	6	R PT / MLS	Poor
76	Kumararaj	49	M	S, HCh,CAD	120/76	7	94	-	L CG	Good

No	NAME	Age	Sex	Past History	BP	NIHss	SU	Hb A1c	CT BRAIN	Prognosis
77	Asia Akthar	50	F	Ht	170/92	10	123	6.1	R FP / MLS	Poor
78	Andal	60	F	RHD, AF	104/68	7	86	-	L F	Good
79	Muthu	72	M	Ht	160/92	7	89	-	L CG	Good
80	Narayanasamy	50	M	Ht, S, HCh	172/100	25	284	10.4	R FP / MLS	Death
81	Solapathy	60	M	Ht	190/110	7	100	-	R CG	Good
82	Gurusamy	65	M	Ht, S, A	200/106	25	104	-	L FPH / MLS	Death
83	Sankar	48	M	Ht, S, A	180/110	4	89	-	L F	Good
84	Sethu	54	M	S	120/80	9	117	6	L F / VS	Moderate
85	Arumugam	54	M	Ht, Dm, S, A	190/100	10	190	-	R PH	Moderate
86	Balakrishnan	82	M	Ht,HCh,CAD	160/110	24	100	-	L FP / VS	Death
87	Ramasamy	60	M	Ht, Dm	190/96	10	188	-	R PH	Moderate
88	Anthony Doss	80	M	Ht	200/110	25	101	-	R PT / MLS	Death
89	Raghu	54	M	Ht, S, A	180/92	7	89	-	L PT	Good
90	Balakrishnan	60	M	Ht, S, A	200/116	10	100	-	L CH	Moderate
91	Ramayee	52	F	Ht	170/90	17	124	6	L PT	Poor
92	Fathima	60	F	Ht	170/90	4	93	-	L CG	Good
93	Ismail	72	M	-	120/80	7	90	-	R CG	Good
94	Pitchai Saibu	65	M	S, A	130/82	14	120	6.1	L PT / MLS	Poor
95	Rajamani	65	F	Ht, CAD	176/100	14	198	10.4	R FH / VS	Moderate

No	NAME	Age	Sex	Past History	BP	NIHss	SU	Hb A1c	CT BRAIN	Prognosis
96	Ramu	60	M	S, HCh	120/84	7	97	-	L F	Good
97	Periyanagammal	70	F	Ht	160/90	8	124	6.1	R FH / VS	Moderate
98	Abdul Rahim	60	M	S, A, HCh	120/84	7	88	-	L FP	Moderate
99	Neelkandan	58	M	Ht, S	210/110	23	240	6.1	L FPH / MLS	Death
100	Kottaya	78	M	Ht, S, A	200/100	25	106	-	R FPH / MLS	Death

HT-Hypertension ; DM – Diabetes ; S – Smoker ; A – Alcohol ; CAD - Coronary artery disease ;

AF – Atrial fibrillation ; HCh - Hyper Cholesterolemia ; SU – Admission day blood glucose level ;

RHD – Rheumatic heart disease ; MVP – Mitral valve prolapse ; DCMP – Dilated cardiomyopathy ;

Hb A1c – Glycosylated hemoglobin ; L – left ; R – right ; F – frontal infarct; FP – fronto parietal infarct;

PT – Parieto temporal infarct; THA – Thalamic infarct ; CEREBELAR – cerebellar infarct ;

FH – Frontal hemorrhage ; FPH – fronto parietal hemorrhage ; PH – parietal hemorrhage ;

PTH – parito temporal hemorrhage ; CH – capsulo ganglion hemorrhage ; CG – capsule ganglion infarct ;

VS – ventricular squashing ; MLS – midline shift.